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THE EFFECTS OF CARBON MONOXIDE POISONING AND HBO THERAPY ON FIBRONECTIN

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ABSTRACT

We used immunological rocket electrophoresis for quantitative testing of the plasma fibronectin (PFn) of 35 cases of acute carbon monoxide poisoning and following hyperbaric oxygen (HBO) treatments. The results indicate that with carbon monoxide poisoning, there is a marked drop in PFn, a very significant difference from the control group. Following one week of HBO therapy, the patients' PFn returned to normal levels. This indicates that HBO therapy is of major significance in promoting the active recovery of fibronectin (Fn).

KEY WORDS: Fibronectin, carbon monoxide poisoning, hyperbaric oxygen.

Fibronectin is a group of high molecular glycoprotiens which are structurally similar and immunologically the same. There are plasma and cellular fibronectins. Fibronectins are synthesized by the skin cells within the blood vessels, muscle cells, fibrous cells and epidermal cells. They mainly exist on the surface of human and animal cells and in the blood plasma. Fibronectin performs a variety of biological activities. Its concentration in the bodily fluids is of major clinical significance[1-3]. We observed the plasma fibronectin of a portion of acute carbon monoxide poisoning cases treated in our hospital and the dynamic changes in the fibronectin during the process of hyperbaric oxygen treatments. The results of these observations are reported below:

SUBJECTS AND METHODS

I. OBSERVATION OF SUBJECTS

For the patient group we selected 35 cases of carbon monoxide poisoning who all had been immediately sent to the hospital for emergency treatment. Of these 35 cases, 15 cases were of moderate degree of poisoning and 20 cases were of acute poisoning. The average age of the

patients was 44 years old. Prior to the poisoning, all were in good health with the exception of one patient who suffered from high blood pressure. The control group was made up of 32 healthy persons who also entered the hyperbaric oxygen chamber.

The patient group was divided into four groups, the moderate poisoning before and after and acute poisoning before and after groups. We observed the plasma fibronectin content of these groups, and we conducted plasma PreA, CK, T_3 and T_4 tests prior to and following treatment.

II. METHODS

When the patients were admitted to the hospital for emergency treatment, they received hyperbaric oxygen therapy. After one week, blood was drawn from their veins early in the morning. All the blood for each sample was drawn at one time, and at that time was divided into two tubes, one for anti clotting (0.2 milliliters of 3.38 percent sodium citrate was added to 1.8 milliliter of blood) and non anti gelling tubes. These were quickly separated into plasma and serum and the various tests were performed. The remainder of the samples were stored at -20xC. When these were retested, the frozen samples were melted by placing them in a 37xC water bath for 15 minutes.

The patients with moderate poisoning were given HBO treatments of two atmospheres (ATA) to breathe pure oxygen for 30 minutes on two occasions. Each; treatment lasted two hours, and was given once a day. The patients suffering from acute carbon monoxide poisoning were given 2.5 ATA, and were placed in the chamber to breath pure oxygen on three occasions, for 30 minutes on each occasion. Each treatment process for these cases lasted 2.5 to three hours, and was given from two to three times per day. When the patient started to improve, he was switched over to conventional treatments.

Immunological rocket electrophoresis was used for the Fn and PreA tests. The antiserum and standard samples were provided by the Shanghai Biological Products Institute. Creatine coloring methods were used the CK testing. Isotope marking methods were used for the T_3 and T_4 tests.

RESULTS

Table one shows the comparison of the plasma Fn content of the moderate and acute carbon monoxide poisoning patients before and after HBO therapy with the normal group. We can see from this table that prior to therapy, there was a very significant difference in the plasma Fn content of all those suffering from moderate and acute poisoning and the plasma Fn content of the normal group. However, following treatment, the plasma Fn content of the patient was not significantly different from that of the control group.

(1) 组 别	(2) n	Fn(mg/L)	(3) P值
(4) 中度中毒治疗前	15	141±44	<0.001
(5) 中度中毒治疗后	12	289±74	>0.05
(6) 重度中毒治疗前	20	64±28	<0.001
(7) 重度中毒治疗后	17	278±93	>0.05
(8) 正常对照组	32	317±86	

COMPARISON OF PLASMA FN CONTENT OF CARBON MONOXIDE PATIENTS BEFORE AND AFTER TREATMENT WITH THAT OF NORMAL GROUP

1. Group. 2. Number. 3. P value. 4. Intermediate poisoning before treatment group. 5. Intermediate poisoning after treatment group. 6. Acute poisoning before treatment group. 7. Acute poisoning after treatment group. 8. Normal control group.

The changes in the plasma Fn and the serum PreA, T_3 and T_4 content are shown in table two. In carbon monoxide poisoning cases, prior to treatment there is a clear negative correlation between the plasma Fn and the serum CK activity peak ($n = 31$, $r = 0.60$, $P < 0.01$). Following hyperbaric oxygen treatments, as the plasma Fn rose, the CK also quickly returned to normal. There was no clear correlation between the plasma Fn content and the preA, T_3 of T_4 either before or after treatment.

(1) 组 别	Fn		PreA		T_3		T_4	
	n	mg/L	n	mg/L	n	nmol/L	n	nmol/L
(2) 治疗前	35	97±52**▲	35	340±82	20	2.6±0.8	20	126±28
(3) 治疗后	29	283±84*	29	375±63	20	2.6±0.8	20	123±26

TABLE TWO: BLOOD FN, PRE-A, T_3 AND T_4 LEVELS OF CARBON MONOXIDE PATIENTS BEFORE AND AFTER TREATMENT

1. Group. 2. Before testing. 3. After testing.
Note: Comparisons of the two groups. * $P < 0.001$. compared to normal control group delta $P < 0.001$.

For one week we observed the serum Fn content of the carbon monoxide poisoning patients and the normal control group prior to each HBO treatment and after coming out of the HBO chamber. The results are shown in table three:

(1) 组 别	(3) 时 间 (天)						
	1	2	3	4	5	6	7
(4)中毒者(5)进仓前	43.7	140.0	161.5	204.0	250.0	316.1	338.0
(6)出仓后	171.8	203.1	260.0	289.0	378.1	381.1	349.3
(7)健康者(8)进仓前	289.7	301.3	356.2	410.0	400.0	425.0	400.0
(9)出仓后	464.7	482.3	482.3	512.1	450.0	415.0	420.0

TABLE THREE: DYNAMIC CHANGES (MG/L) IN PLASMA Fn CONTENT OF CARBON MONOXIDE PATIENTS AND HEALTHY PERSONS BEFORE AND AFTER HBO

1. Group. 3. Time (in days). 4. Carbon monoxide poisoning patients. 5. Prior to entering HBO chamber. 6. After leaving chamber. 7. Healthy group. 8. Prior to entering HBO chamber. 9. After leaving chamber.

DISCUSSION

Current research indicates that patients with septicemia, serious injury or kidney disease all display a decrease in the plasma Fn level to varying degrees. Saba and others[1] believe that the drop in plasma Fn level during septicemia is due to the formation of collagen fragments, platelet damage, and the formation of immunological compounds, germ or non germ granules during the transitional process of the disease, and in the process of the Fn clearing these products out of the endothelium in its regulating role, it is used up. We also tested the serum CK activity, as an objective index of the degree of damage to the tissue cells in the reacting body. After carbon monoxide poisoning, there was a marked rise. Its peak showed a markedly negative correlation to the plasma Fn content. This indicates that a drop in plasma Fn content follows tissue cell damage, and the amount it drops is related to the degree of poisoning.

During carbon monoxide poisoning, due to tissue oxygen deficiency, energy metabolism is hindered, and tissue cells are damaged. Furthermore, blood vessel endothelium cells are especially sensitive to oxygen deficiency, and a fairly long period of oxygen deprivation can cause endothelium damage. These factors can all cause a reduction in Fn synthesis, with an increase in its disintegration and consumption. This is the primary cause of the drop in plasma Fn.

We also observed a marked drop in plasma Fn following carbon monoxide poisoning. After one week of HBO therapy, it was restored to approximately normal levels. Prior to and following each HBO treatment, the dynamic changes in the plasma Fn content was observed, and the results indicate that HBO therapy not only clearly affects the Fn levels not only of carbon monoxide poisoning patients, but of healthy individuals as well. After each HBO treatment, the plasma Fn content was markedly higher than it was prior to entering the HBO chamber. Even though this elevated Fn level had dropped some prior to entering the chamber for the next treatment, the overall Fn level showed an elevated trend. As the number of treatments increased, the difference in the plasma Fn content prior to

and after entering the HBO chamber gradually decreased. After one week, it tended to level out, causing the plasma Fn content to be maintained at a relatively higher level. This result indicates that HBO has a marked effect on human Fn content, and that HBO therapy may be of major significance in promoting the Fn content level to recover on its own. However, further exploration will be required to determine the mechanism for this.

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